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PRINCIPAL FORMS OF INTRACRANIAL HYPOTENSION

Second Report

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16. Abstract

After a short historical survey and a discussion of some anatomical-surgical considerations, we discuss in detail the clinical aspects of intercranial hypertension. We discuss the synthesis of physiopathological concepts now known which make it possible to carry out an interpretation test of the syndrome.

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PRINCIPAL FORMS OF INTRACRANIAL HYPOTENSION

P. Puech, P. Guilly, J. Morice, and M. Brun

In a series of 238 cases of hypotension, confirmed by surgery, it seemed interesting to study the main forms of the important syndrome of intracranial hypotension, which is sometimes also associated with other malfunctions of the cerebromeningeal hydrodynamics. /458*

I. History

The traumatic intracranial hypotension was characterized by Professor Leriche during the 1914 — 1918 war, who also precised the pathologic physiology and the treatment. The role of the choroid plexus was brought to attention in 1920 by Henschen. /459 Hoseman (1921) records 100 cases of hypotension after rachicentesis. Puech, Rappoport, and Brun (1935) report observing the cure of an intraventricular hemorrhage with hypotension after "washing and reinflation of the ventricles." Many neurosurgeons comment on accidents of postoperative ventricular depletion. David, Berdet, Mahoudeau, and Askenasy (1935) show the importance of hypotension in the pathology of certain postoperative cerebral hemorrhages. Mahoudeau (1936), in his very important thesis on postoperative hypotensions, brings to light the preponderance of hypothalamic dysfunctions. It is to prevent such postoperative accidental depletions that de Martel and Guillaume (1937) recommend the preoperative drainage of the ventricular cavity, and that one of us, at the end of the surgery, after

*Numbers in the margin indicate pagination of the original foreign text.

cutting away all large introcranial neoformation, or concomitant to a sudden release of the cerebrospinal circulation, systematically re-establishes, since 1934, a normal intraventricular pressure by directly injecting air or serum into the ventricles. About the same time, M. Schaltenbrand reports very similar facts; discussing the rule of hypotension in subdural hematoma (1935), he insists on the spontaneous hypotension (1938) which he calls very suggestively "aliquorrhee" (absence of fluid); H. Wolff (1942) mentions three observations. Thiebaut (1945) describes hypotension in the different forms of ventricular dilatation. In many publications, the first one already in 1935, one of us studies the hypotension secondary to many medical and surgical affections and the primary clinical forms of this hypotension.

II. Frequency

The well-confirmed intracranial hypotension represents about 7.2% of the cerebral neurosurgery in our statistics; 238 cases of hypotension are recorded among 3,284 intracranial surgeries in the Neurosurgery Department of the Hospital Sainte-Anne. In those cases, the hypotension is tested by trephining and puncture or by direct cerebral exploration. We did not retain the cases where the diagnosis was revealed by rachicentesis and by sub-occipital puncture: very often these cases do not show what is actually going on in the ventricles, and the idea of a pressure uniformly modified in all of the cerebrospinal system, much too simplistic, must be reviewed.

We consider then mainly the neurosurgical forms of intracranial hypotension which justify a "re-inflation of the ventricles." The common cases cured by a "cerebromeningeal rehydration" are more frequent than our statistics would allow to suppose.

Among the 238 cases of intracranial hypotension, 68% are secondary and 32% apparently primary. Among the secondary forms, 18.8% occur following accidental trauma, 10.5% are a post operative accident, and 38.7% are secondary to a medical or surgical affection. This means that sometimes the doctor, and some other times the surgeon, has to foresee and prevent, or to detect and treat the intracranial hypotension. The clinical diagnosis is often quite difficult, but it is very important to keep it in mind on many occasions. /460

III. Anatomy

Hypotension expresses itself to the neurosurgeon under two main forms:

— hypotension alone

— hypotension associated with other intracranial disorders.

A. The Most Frequent is the Isolated Hypotension

Among the 238 cases examined, hypotension alone has been found 135 times. This list can be broken down into:

— 24 posttraumatic cases

— 25 postoperative cases

— 19 cases secondary to medical or surgical affections

— 67 cases of apparently primary hypotension.

No matter what the etiology of the isolated hypotension, the neurosurgeon finds the dura understretched and not beating. After incision of this membrane, the brain appears reduced in volume, abnormally far from the skull, collapsed, and without obvious beat. This state of cerebral dehydration is confirmed

by ventricular puncture: no fluid is extracted by penetration of the trocar, and a hissing noise of air aspiration can be detected. If some liquid is still found in the ventricle, the pressure is strongly negative: this liquid can only be withdrawn by aspiration, or by tilting the head backwards, or by compression of the jugular veins. This negative pressure can actually be measured with a manometer.

B. Intracranial Hypotension Is Often Associated with Other Intracranial Disturbances

In our series of 238 cases of confirmed intracranial hypotension, 103 cases of hypotension "associated" have been recorded.

Two different types have to be considered:

— the type where hypotension is associated with other cerebromeningeal hydrodynamics troubles

— the type where hypotension is associated with other intracranial lesions.

a) Hypotension associated with other cerebromeningeal hydrodynamics troubles: 71 cases in the series examined, with 16 traumatic, 46 secondary, and 9 apparently primary.

In these cases, again it is the technique of trephining and puncture which confirms the diagnosis.

With hypotension, one observes:

1. A certain level of cerebral edema more or less diffuse (brain in "gelatin" of Clovis Vincent): 7 cases.

2. Or a ventricular dilatation in spite of a hypotension /461
of the fluid (Puech, Buvat, Koechlin, and Brun, 1942; Thiebaut, 1945, 1946, 1947): 38 cases.

3. Or a serious meningitis or subdural hygroma (Krebs, Puech, and Brunhes, 1937): 26 cases.

The cerebral mass and the volume that it occupies in the skull are large enough to conceive the possibility of such associations, confirmed by surgery.

One or the other of the cerebromeningeal hydrodynamics trouble can be primary or secondary.

Classically, these reactions are seen as complications of large lesions such as tumors, abscesses, posttraumatic lesions.

The reprecussion of a whole series of medical or surgical affections is also known, where an infection or intoxication can complicate the problems, sometimes lethal, of the cerebromeningeal hydraulics.

At last, this trouble appears often as primary clinical signs. They must be searched for systematically by exploratory trephining and puncture procedure in a series of serious or persistent neuropsychic syndromes whose etiology is not obvious.

Though cerebral edema is usually given a preponderant role, we tried to demonstrate that the various other troubles of cerebromeningeal hydraulics are as important. In a series of 949 troubles predominantly of the cerebromeningeal hydrodynamics of various etiology, we observed ventricular hydrocephaly in 24.9% of the cases; serous meningitis in 36.6%; cerebral

edema in 20.8%, and hypotension in 17.7%: in a third of those cases, the various troubles were successive or more or less associated.

949 CASES OF MALFUNCTIONS OF THE CEREBROMENINGEAL HYDRODYNAMICS CONFIRMED BY SURGERY

Most important trouble	Origin		Total	Frequency %
	Posttraumatic and postoperative	Nontraumatic		
Ventricular dilatation with or without hypertension	43	195	237	24.9
Serous meningitis	125	221	346	36.6
Cerebral edema	36	162	198	20.8
Hypotension	67	101	168	17.7

It seems to us that the signs of "localization" observed frequently (localized paralysis, B-J epilepsy, localized headaches, electro-encephalographic disturbances) are in direct relationship either with a cerebral collapse particularly marked in the area corresponding to the localized signs, or with a localized serous meningitis; or again with a localized cerebral edema associated with hypotension. /462

It is important to recognize these different forms and to distinguish them from the cerebral collapse bound to a direct compression of the brain. The latter is concomitant to intracranial hypotension. The pressure can be due to a sub-dural hematoma or a predominant serous meningitis, so that the hypotension would only be detected after excision of the compressive agent.

b) Hypotension associated with other intracranial lesions: 32 cases

It might be posttraumatic lesions, or nontraumatic lesions diffuse or localized, tumorous, vascular, atrophic, infectious (R. Debre, Thieffry, and Doumic, 1947), toxic. All of those lesions can be comitant to other troubles of the cerebromeningeal hydrodynamics. J. Lebeau (1947) shows that a temporal hernia can be associated with intracranial hypotension or hypertension. In any case, the primary lesion must be treated first; then, if necessary, the troubles of the hydrodynamics must be looked after.

IV. Clinical Signs

Intracranial hypotension bound to a more or less cerebromeningeal dehydration can present variable clinical pictures, sometimes even close to that of intracranial hypertension. In both cases, the patients complain of headaches and vomiting. In 15% of the cases, they can also present a slight but unquestionable papillary edema: it is the result of our experience, though other authors reported that the papilla stays normal during hypotension. In both cases, electroencephalographic patterns can be changed in a diffuse, or more or less localized way.

Three main clinical aspects can represent the intracranial hypotension: neurological, psychic, and neuropsychic; the frequency of each group of disorders being practically equal (see table below relating the clinical syndromes most frequently observed in our series):

Neurological Forms

Sometimes the patients give a picture of cerebral apoplexy more or less complete: coma with or without hemiplegia, which points towards the diagnosis of cerebral softening or cerebromeningeal hemorrhage (Puech, Rappoport, and Brun, 1935). At other times, the patients first show an appearance of deficiency with hemiplegia, monoplegia, and ocular paralysis (Puech, Perrin, and Koechlin, 1942). In other cases, the patient is brought to us because of general or localized convulsions, or because of a subintrant crisis ("etat de mal") (Leriche and Wertheimer, 1925). They show the usual hypothalamic signs (Mahoudeau, 1936): the amplification of those reveals a true hypothalamic form of the disease. /463

PREVAILING CLINICAL SYNDROMES IN 204 CASES OF INTRACRANIAL HYPOTENSION

Syndrome*	Nontraumatic	Traumatic	Total
Epilepsy	61	5	66
Coma	18	14	32
Confusion	4	16	20
Melancholic depression	19	2	21
Various paralysis	11	1	12
State of intellectual debility	9	2	11
Insipid diabetic and narcolepsy	9	1	10
Schizophrenia	5	2	7
Aphasia	4	1	5
Various neuropsychiatric syndromes	19	1	20

*Clinical signs of hypothalamic dysfunction (hyposomnia and disturbance of the thymic state, polyuria, general dehydration, vasomotor troubles) are present in each case.

The following observations describe the main forms of neurological intracranial hypotension.

1. *With cerebral apoplexy.* M.L., 46 years old, professor, referred to emergency by Drs. Ortolan and Hallot, because of coma with hemiplegia. The diagnosis made was softening of the brain but the observation of papillar edema along with progressive deterioration of the general condition of the patient incite to refer to a neurosurgeon's opinion.

In the medical history of this patient, one records for the previous year, five times, when fasting in the morning, a state of euphoria with hypomania, and some attacks of narcolepsy.

Trephining and puncture reveal hypotension: 40 cc of air were injected. Then the recovery was rapid. For eight years, M.L. has been back to teaching.

2. *Meningeal form* (tuberculous pseudomeningitis). Mrs. H., 45 years old, living in Brest from where Dr. Baumgartner calls us in emergency, in July 1933, for a case of coma with meningeal syndrome.

One month previously, there was a brutal attack of headache and ictus during a walk. Meningeal signs with arterial tension elevated to 18 during the ictus crisis (14, normally). Hemorrhagic lumbar puncture.

The meningeal symptoms persist and lead to a state of coma with Cheynes Stockes' respiration. In the eye ground, the papillary edges are diffuse and the veins dilated. The general condition is very poor, and in spite of the absence of Koch Bacillus in the cephalo-spinal fluid, the clinical signs are those of a tuberculous meningitis.

Puncture of the ventricle at the third day of coma: important hypotension, re-inflation of the ventricles. The patient has been cured for fifteen years.

Miss V.,* 23 years old, referred on June 4, 1948, by Dr. Deparis, for a meningeal syndrome with torpor and occular signs leading to a tentative diagnosis of turberculous meningitis to be treated with streptomycin. /464

In 1945, pulmonary attack, followed by serofibrinogenous pleurisy with a loss of weight of 17 kg.

In May, 1948, occipital headaches on the left side, occuring in crisis more and more frequent.

On the 14th of June, 1948, a meningeal syndrome appears with excruciating headaches, vomiting, and photophobia. A state of somnolence and a slight mental confusion persist. Medical examination shows a left brachial monoplegia, a paresis of the VII left, as well as diplopia, bilateral mydriasis, and bilateral ptosis. Also present are a lateral nystagmus in the horizontal meridian, and a paroxystic hiccup. Strong peripheral excitations bring about a posture in opisthotonos.

This clinical picture hints to a tuberculous meningitis or possibly a tubercle; a ventriculography is performed which reveals an intracranial hypotension.

A rapid improvement comes along after restoration of a normal ventricular pressure. The patient leaves cured on the 26th of June, 1948.

*This fact was observed later, after the meeting of our two societies.

3. *Hemiplegic form.* Mrs. B., 36 years old, nurse, referred by Boyer Chassigneux on May 6, 1946, for a left hemiplegia.

The motor troubles started one month previously, fifteen days after surgery for appendicitis and retroversion of the uterus. Left hemiplegia with headaches, depressed state, and some character disturbances. The eyes ground is normal. Arterial tension: 15/9. Trephining and puncture: hypotension without ventricular dilatation. Ventricular fluid normal. Re-inflation. Cured for two years.

4. *Pseudo-tumoral form.* Mrs. V., 38 years old, referred by Dr. Brunel, October 15, 1947, for a syndrome of headaches with slight papillary edema.

Arterial tension: 12/6.

Ventriculography shows a strong hypotension. Ventricular fluid: albumin 0.12 g; 12 elements. Restoration of normal pressure brings about cure.

5. *Ocular form.* Mrs. P., 24 years old, referred by Dr. Kalt, September 3, 1943, for an optochiasmatic arachnoiditis.

The vision is improved after cure of the arachnoiditis. The patient comes back in May, 1944, for a blindness of rapid onslaught. It seems like a relapse, but a preoperative ventriculagraphy shows a strong hypotension of the ventricles, not distorted.

Re-inflation of the ventricles brings about a rapid return of eyesight.

This case demonstrates an ocular form of intracranial hypotension with blindness, but it can exist also with papillary edema or with disturbances of the ocular motility.

6. *Form with convulsions.* They can be represented by a subintrant crisis, general or localized epilepsy.

a) Type subintrant crisis ("etat de mal"). M.S., 17 years old, referred by Professor Delay, on October 1, 1947, for an epileptic state.

Epilepsy, of the general type, started when the patient was 8 years old. Repetition of the crisis in spite of medical treatment, which progressively developed into psychomotor equivalents (depression and two attempts at suicide).

A subintrant crisis occurs suddenly. Examination of the eye ground shows that the papilla are slightly dulled.

Ventricular puncture: hypotension.

Cure for seven months following re-inflation, but on May 16, 1957, brutal onslaught of a new "etat de mal."

Ventricular pressure in emergency: re-inflation of the ventricles; the crisis ceases.

b) Type general epilepsy. Mrs. C., 35 years old, /465
referred by Dr. Lereboullet on April 9, 1946, for epilepsy which appeared for the first time at age 19. For several previous months, the crisës are more frequent, with headaches, vomiting, fogginess in front of the eyes. Eye ground: pale papilla, vision 3/10.

Ventricular puncture: hypotension with normal ventricular fluid. Simple re-inflation.

c) Type with B-J epilepsy. Mrs. R., 65 years old, referred by Dr. Chapoulaud on June 22, 1946, for epileptic crisis which started 5 years previously: first, general crisis, later, right brachial B-Jacksonian type of epilepsy.

Ventriculography: hypotension with asymmetrical ventricular dilatation (left atrophy). Ventricular fluid normal.

After simple injection of air, cure of the crisis for two years.

Psychic Forms

We describe a type with melancholic depression (Puech, Bessiere, Micoud, and Brisson, 1942), a type with mental confusion (Delay, Puech, and Maillard, 1945), a type with periodic mania, and a form with narcolepsy (Puech, Perrin, Koechlin, 1942; Bessiere, Puech, and Morice, 1945). Thiebaut and Daum (1945) have described a form simulating general paralysis. We also observed some forms presenting certain schizophrenic type of states.

Considering this documentation, two theses have been brought forth:\ the thesis of M. Morice (1944), Assistant at the Neurosurgical Center of Sainte-Anne, and the thesis of M. Sizaret (1947), a student under Professor Delay. For several years, M. Delay insists on the action of the "Cerebral Pneumotherapy" in intracranial hypotension; but he emphasizes also the important role played by pneumotherapy in other

psychiatric syndromes, with action on the noetic domain on one hand, and on the thymic area on the other hand. Guiraud and Morice (1946) emphasize also the value of lumbar injections of air in psychiatric therapeutics.

We will also mention a few observations of psychic form of intracranial hypotension.

1. *Type with melancholic depression.* M.M., 47 years old, referred by Dr. Bessiere on October 8, 1940, was committed to a mental institution for melancholic depression with suicidal and indignity tendencies.

Ten years previously, first episode of melancholia requiring commitment. Then the depressive state worsens, with grave asthenia and temporary intellectual deficiency. Left B-J epileptic attacks, then left hemiparesis following rachicentesis.

Trephining and puncture: hypotension. Re-inflation. Cure for seven years of the neurologic and psychic signs.

2. *Type with mania.* Mrs. S., 39 years of age, referred by Professor Delay, April 13, 1946, for typical manic state with euphoria, temporary loss of thoughts logorrhea, familiarity crisis, motor hyperactivity.

Three previous crises treated by electroshock therapy.

Amplification of the troubles. Insomnia, loss of weight, polydipsia. Eye ground normal.

Trephining and puncture: hypotension; after restoration of a normal ventricular pressure, improvement of the hyperactivity and of the loss of thoughts; however, the troubles of

the manic series did not disappear completely.

3. *Confusion form.* Mrs. F., 30 years old, referred by Professor Delay. Admitted in the Department of the Clinic at Sainte-Anne for the first time on May 27, 1943, for grave confusional state with, alternatively, adynamia and hyperactivity. Temperature fluctuating between 39 and 40° C.

Blood urea: 0.99 g; albumin: traces in urine. /466

Lumbar puncture: alumbin 0.40 g, 5 elements. Hemoculture: negative.

After the usual therapeutics are shown to be inefficient, the patient receives some electroshocks. She seems cured and leaves on July 1, 1943, to lead a normal life.

One and a half years later, the patient comes back to the Clinic for a new confusional crisis. Temperature 38° C, blood urea 0.88 g. Cure after a second series of electroshocks.

On April 19, 1945, third confusional episode with temperature 39.5° C, blood urea 1.5 g, albumin 0.82 g. Lumbar puncture: albumin, 0.40 g, 4 elements; Meinicke, Pandy and Benjoin tests: normal. After another series of electroshocks, retrogression of all the symptoms. Fourth relapse on May 24, with confusion and hyperactivity, fever, and urea 0.50 g. Eye ground normal.

Puncture: complete absence of fluid. Ventricles: normal. Re-inflation of ventricles by air, and cure maintained up to now.

4. *Schizophrenic form.* (after recent closed traumatism). M.L., 22 years old, referred by Professor Aubertin, on August 31, 1944, following a right frontal cranio-cerebral traumatism five days previously.

After a brief confusional period, a schizophrenic state takes place with catatonic attitude, negativism, opposition, total lack of interest, dissension, giggling, and puerilism.

Left hemiparesis with bilateral Bâbinsky's sign is recorded also. Pulse slowed down to 46. Slight papillary edema.

Trephining and puncture: hypotension. Re-inflation of the ventricles. Cure for four years.

The EEG recording, disturbed in many areas but mainly in the frontal region, became normal again two months later.

5. *Form with type "general pseudo-paralysis."* M.L., 58 years old, referred by Dr. Senges on December 24, 1946, for a frontal syndrome with euphoria, disturbances of the memory and judgment, changes in the behavior and indifference reminding of the aspect of P.G. (general paralysis).

Eye ground normal, with narrowing of the visual field.

Trephining and puncture: hypotension, with some ventricular dilatation of the lateral ventricles but important dilation of the IIId ventricle.

Ventricular fluid: albumin: 0.15 g, 0.2 elements; Benjoin: normal. EEG recording flat, no abnormality.

Temporary improvement after re-inflation.

6. *Form with "type narcoleptic."* Mr. R., 46 years old, referred by Dr. Monier-Vinard on May 13, 1943, for narcoleptic outbursts starting in 1936, occurring during work, and lasting sometimes several days.

More recently, apparition of polyuria, with psychic troubles characterized mainly by intellectual weakening. Eye ground normal.

Ventriculography June 25, 1942. Slight ventricular dilatation without neoformation. After suppression of the grave hypotension, an immediate improvement occurs, which lasts since 1943.

Neuropsychic Forms

The intracranial hypotension is often expressed by association at different degrees of neurological and psychic signs.

V. Evolutional Forms

Intracranial hypotension has either an acute or superacute evolution (Delaunay and Demarez); or a superacute or chronic evolution such as the cases we emphasized. Acute or chronic, the evolution is generally schematic; the preamble period which can last from a few days to a few months is marked by headaches, loss of weight, modification of the thymic state, and often by a diabetes insipidus more or less pronounced. The symptoms of /467
this phase are often only found later on, retrospectively, during the period of "state," when grave neurological or psychic troubles have appeared. Those begin sometimes insidiously and sometimes brutally after a strong onslaught of headaches often fronto-temporal, localized, with irradiation towards the face,

so that one can believe it a facial neuralgia. The questionning reveals then that the first symptoms occurred mostly in the morning, far from mealtimes, and were reduced by ingestion of food or drink, or by a decubital posture with head lowered. If a "re-inflation of the ventricles" is not performed rapidly, the patient falls into coma and dies.

In some other cases, the hypotension persists for months or years at a chronic state: the diagnosis is erroneous up to the day where a ventricular puncture discovers a ventricular collaps similar to Clovis Vincent's.

VI. Etiological Forms

To discover the cause of the hypotension is sometimes easy, but often very difficult. There are secondary forms, and forms clinically primary.

238 CASES OF INTRACRANIAL HYPOTENSION CONFIRMED IN A SERIES OF 3284 OPERATIONS OF INTRACRANIAL LESIONS

Cause for hypotension	Number of Cases	Frequency
Posttraumatic: 70 cases		
1. accidental	45	18.8%
2. operative	25	10.5%
Nontraumatic: 168 cases		
1. secondary	87	36.5%
2. therapeutic	5	2.1%
3. primary	76	32.1%

In these statistics are included: the predominant hypotension (110 cases), i.e., 462%; the cases where hypotension is associated with a serous meningitis (26 cases), with ventricular dilatation (38 cases), with cerebral edema (7 cases), i.e., 29.8%; and the cases where it is associated with other intracranial lesions (57 cases), i.e., 24.5%.

The study of the medical history, the clinical examination, the investigation of biological signs of hypotension, and particularly the systematic study of the ventricular fluid, with often ventricular hemorrhage, hyperalbuminosis, and sometimes, on the contrary, a predominantly leukocytic reaction; the comparison of the fluid of serous meningitis and of the ventricle contents; a complete humoral study, the decrease of the ratio serine/globulin (Delay and Sizaret) bring sometimes useful information.

1. *Posttraumatic hypotensions*. Posttraumatic hypotension is known by surgeons since the work of Leriche.

Posttraumatic hypotension, in our study, represents 18.8% of all the hypotension: 45 cases out of 238. It is often caused by recent accidents (37 cases), more rarely of late accidents.

In both cases, it can be either an open cerebral traumatism with expulsion of the fluid by the ears and nose, or more often, of a closed cerebral traumatism.

Along with Krebs, we reported a number of observations of posttraumatic hypotension, and we would like to underline that one of the preceding observations, expressing a state of post-traumatic schizophrenia, was done on a recent closed traumatism.

This second case seemed particularly interesting to us, featuring a transfixing cerebral wound by bullet.

M.D., 20 years old, referred by Dr. Bauzet on January 7, 1944, for a transfixing wound by bullet: penetration right sub-zygomatic exit right paramedian occipital. The bullet is under the skin.

Patient in complete coma.

Emergency operation on January 7, 1944.

1) Operation on the exit orifice: amputation of the right occipital pole.

2) Operation on the entrance orifice.

The next day, polyuria, diabetes insipidus, treatment by hypophysis powder.

On January 20: persistance of the diabetes insipidus with motor agitation. A depressed scar is observed, and the wound is rehydrated.

February 15: hypophysial cachexia. The patient is still in the coma.

February 21: puncture of the ventricle which shows a marked hypotension.

Re-inflation of the ventricle. Blood transfusions; progressive improvement.

March 15: still improving. Disappearance of the diabetes insipidus. The patient is lucid enough for us to observe a lateral homonymous hemianopsia on the left side with alexia.

May 15: the patient is released; lateral homonymous hemianopsia and alexia are persistent. EEG: slow waves with right occipital derivation.

July 9, 1946: the alexia has disappeared, persistence of hemianopsia. EEG normal.

The patient leads a normal life.

2. *Postoperative hypotensions.* They are known since the work of Mahoudeau in the Department of Cl. Vincent.

In our series, we observed 25 cases of this type, i.e., 10.5%.

It concerns various intracranial neoformations. Like Mahoudeau, we have observed some intracranial hypotension in the tumors of the posterior cranial fossa in the child, but also in the adult, and in other types of tumors as well (10 cases among 1017 tumors operated on, and 15 cases in a series of 71 sub-dural hematoma).

Let us remember that it is to prevent postoperative accidents of depletion that deMartel and Guillaume recommend the preoperative drainage of the ventricles.

3. *Posttherapeutic hypotensions.* We will not stress the hypotension following lumbar puncture, rachio-anesthesia, ventricular puncture, ventricular drainage in the hydrocephalus: /469 they are well known and can be associated with other troubles of the cerebromeningeal hydrodynamics.

However, one can observe post-radiotherapeutic hypotension, and also after various therapies such as sulfamides, penicillin, or after diabetic coma.

Here are two observations: the first relates to a case of hypotension after radiotherapy (as already reported by Stuhl, Berdet, and Thiebaut in 1938), the other showing a hypotension consecutive to insulinic coma.

Postradiotherapy hypotension. M.F., 30 years old, referred by Drs. Stuhl and Moser, on May 15, 1943.

This patient had a first operation in 1943 because of B-J epilepsy crisis, which revealed an angioma on the left motor area, and received 2850 r.

In May, 1943, new crisis and beginning of a left hemiplegia. The operation allows the removal of a large glioblastoma.

Radiotherapy in November, 1943: 9000 r. Improvement.

February, 1944: the patient gets worse.

March 16, 1944: new operation which shows a very hypotensive brain but no trace of tumor.

The troubles disappear after rehydration.

Postinsulinic hypotension. Miss L., 32 years old, referred by Professor Delay in November, 1947, because of subintrant crisis which occurred at the end of an insulinic coma, five minutes after IV injection of 150 cc of glucosed serum at 30%.

It was a case of precocious insanity; committed for the fourth time and on whom electroshocks had no results.

Ventricular puncture on November 24, 1947: ventricular hypotension without dilatation. Re-inflation. Immediate disappearance of crisis and coma. The psychic syndrome is not modified.

4. *Hypotensions secondary to multiple medical and surgical affections.* A hypotension secondary to various medical and surgical affections is discovered by practitioners and surgeons: it was the case 87 times in our series of 238 cases of hypotension.

Among those 87 cases of secondary hypotension, one records:

— 6 cases of encephalitis with pseudotumoral aspect;

— 5 cases consecutive to an infection of the cavities of the face, reminding of meningitis or intracranial abscess;

— 4 cases of acute meningitis;

— 1 tuberculous meningitis;

— 3 cases of general paralysis;

— 1 acute azotemic delirium;

— 4 cases of genito-urinary affections;

— 5 cases of ventricular hemorrhage;

— 18 cases of cortical atrophy;

— 28 cases of various ventricular dilatations;

— 12 cases of exogeneous or endogeneous toxi-infections.

It is not possible to report all of those observations, but we will give a few demonstrative examples.

1) *Meningeal postotitic form.* L. Gerard, 13 years old, referred by Dr. Cocheme, on March 27, 1948, with a meningeal syndrome with headaches, vomiting, photobia. Examination of the fluid shows an aseptic meningitis with 4950 elements and 1.20 g albumin.

Eye ground normal.

This young boy was carrier of a chronic left otitis, the /470
clinical diagnosis being very likely of abscess or meningitis. Operative diagnosis: hypotension. Ventricular fluid: albumin 0.22 g, 6 elements. Re-inflation and cure.

2) *Forms secondary to cerebrospinal meningitis.* Child C., 4 years old, referred by Professor Debre and Dr. Thieffry, for a cerebrospinal meningitis resistant to sulfamide treatment.

After a temporary improvement after a treatment by 1162F, hyperthermic coma with sterile fluid at the rachicentesis, hinting to a cloisonment.

Ventricular puncture: hypotension. A few drops of sterile fluid. Injection of 35 cc of air.

The child came out of the coma, and recovered after a difficult convalescence.

3) *Form with chronic meningo-encephalitis.* Mrs. C., 39 years old, referred by Dr. Guiraud, on December 20, 1948, because of headaches and vomiting appearing in a former P.G., having had malaria in 1936, with completely negative serology.

For several years, headaches and vomiting mostly when fasting in the morning, and improved by meals; for which the patient went for cures to Vichy, and even had an operation for the "removal of intestinal adhesions."

Neurologic examination: photomotor reflexes absent. Eye ground normal. Ventricular puncture: hypotension without ventricular dilatation.

Re-inflation and cure.

Relapse in February, 1947. Treated in May and October, 1947, by injections of serum and Pitressin. Cured since then.

4) *Hypotension and ventricular dilatation*. M.K., 43 years old, referred by Professor Delay in December, 1946, because of epilepsia since 1935, following a traumatism, and impulsions of extreme violence.

To this psychomotor epilepsia is added in December, 1936, a state of confusional torpor. Neurologic examination: negative.

Eye ground normal.

Ventricular puncture on December 24, 1946: hypotension with ventricular dilatation.

Immediately great improvement and disappearance of the impulsions.

Later, opening of chiasmatic cisterna. Disappearance of the symptoms.

5) *Forms secondary to a ventricular hemorrhage*. Mrs. D., 36 years old, referred by Dr. Cauhape on April 1, 1935, for epilepsy and right hemiglegia.

Four years before, gunshot in the left frontal area proviking an extradural abscess.

One year previously, a general comitial crisis.

Since three days: progressive somnolence, two general comitial crises, then "subintrant crisis" (59 crises during the night).

Upon examination, right hemiplegia with Kerning sign.

Eye ground: edema.

Ventricular puncture (performed in a state very alarming with artificial respiration): tremendous hypotension. A few drops of pinkish fluid are extracted: lymphocytes 0.9, albumin 0.6 g.

Washing of the ventricles and restoration of a normal pressure.

Ventricles are not dilated.

Complete cure.

We would like to remind here that the hemorrhage can be:

— the cause of hypotension (Krebs and Puech, Schaltenbrand, H. Wolff);

— the consequence of hypotension (Schaltenbrand; David, Berdet, and Mahoudeau).

6) *Form secondary to a renal affection.* This is the observation of a child carrier of a cyst of the urachus:

A. Philippe, 5 years old, referred by Drs. Julien Marie and Morax, on June 29, 1947. This child was operated on when two months old for a cyst of the urachus which grew back and formed an umbilical fistula. Urinary stasis with infection

(colibacilli-staphylococci). /471

Since a few months, diabetes insipidus.

For a few days, somnolence with severe headaches on the left frontotemporal region. Alternative periods of agitation preceding the coma.

Cachexia and dehydaration — shudderings and uncoordinated motions of the face and the limbs.

Right Babinskiy's sign. Slow pulse and troubles of the swallowing reflex.

Injection of serum with glucose and chloride. Increased somnolence and coma.

Blood tests: urea 0.50 g; lowered chloride: chlore in NaCl: 4.78 g; chlore in Cl: 2.90.

Operation on June 30, 1947: Dura looks collapsed, cortex detached from the wall, greyish, brain in "gelatin." Puncture of the ventricles: hypotension. Suction of the fluid: albumin 0.10, lower chlorides: chlore in Cl: 3.86; chlore in NaCl: 6.37.

Re-inflation of the ventricles. Slight ventricular dilatation.

Definite cure, then treatment of the vesicular lesion.

In an adult with adenoma of the prostrate, or during a pyclonephritis, similar syndromes can be observed.

7) *We will finish by relating a peculiar observation of intracranial hypotension during Osler disease treated by penicillin.*

M.S., 60 years old, Osler disease treated and cured by penicillin (Dr. Laporte).

Eighteen months after the cure, appearance of narcolepsy and periods of mental confusion.

A coma with hemiplegia sets in.

Blood test: hypochloruremia.

Clinical diagnosis: metastatic cerebral abscess or hydrodynamics troubles.

Operative diagnosis: hypotension with serous meningitis.

Re-inflation and cure.

5. *Hypotension clinically primary* (76 cases). These primary hypotensions, where no satisfactory etiology can be found, can show all sorts of clinical pictures.

One must think of hypotension in the multiple neurologic, psychic, and neuropsychic syndromes where no etiology can be proven.

The untreated hypotension constitutes a frequent "way of dying" under various circumstances (traumatic, operative, circulatory, respiratory, tonic, infectious, by deficiency). It is sometimes possible for the surgeon to avoid this

eventuality and also the appearance of a hemorrhage or of a secondary cerebral softening. We have observed a number of patients showing a classical picture of cerebral apoplexy, in which we were able to prevent the hematoma by restoring the broken equilibrium of the fluid circulation.

VII. Diagnosis

The clinical diagnosis of hypotension is difficult in most cases. The hypothalamic signs emphasized by Mahoudeau are often most important.

The biological diagnosis is delicate, and interpretation difficult.

The EEG, like the clinical signs, can induce an error of diagnosis. From the research of Puech, Fischgold, and G. C. Lairy-Bounes, it appears that intracranial hypotension alone does not alter the recording. A slow curve can express the cause, the troubles associated with, or the effect. However, no matter what the etiology of hypotension, the same authors insist on the importance in prognosis of the degree of alteration of the recording. /472

In any case, the diagnosis could not be confirmed without the trephining-puncture procedure.

For a long time, one thought that the diagnosis could be made on a single test of rachiomanometry. Practically, this might be satisfactory enough in certain etiological conditions: hypotension consecutive to rachicentesis in patients where one is not suspicious of tumor, accidents of the rachianesthesia, hypotension after trephining. But for cerebral traumatisms or

for interpretation of postoperative follow-ups in neurosurgery, this method must be formally rejected, since it may lead to diagnostic and therapeutic mistakes which can be lethal.

VIII. Prognosis and Results

Two hundred thirty-eight cases of serious intracranial hypotension were operated on with 43 deaths: 18%. There is practically no after-effect in isolated hypotensions, but only in the associated forms.

Reversible and irreversible types of hypotension can exist, for example, such as the recurring forms, and finally, incurable forms bound to atrophic lesions of the choroid plexus (Peuch, Bevat, Lerique, and Perrin, 1942): hypotensions with functional mechanisms and hypotension with lesions of Delay and Sizaret (1947).

IX. Pathologic Physiology

It is easy to imagine: it can be an escape of fluid, a depressed secretion, an excess of resorption. Schaltenbrand and Wolf schematized it in a few lines that we take permission to quote: "The hypotension of the cephalospinal fluid can be due either to a decreased secretion of the fluid with constant resorption; or to an increased resorption with constant secretion. These troubles can occur:

"1. If the rush of blood toward the plexus is weakened or interrupted.

"2. If the secretion of the plexus is decreased by nervous factors.

"3. If the lesions in the plexus cells prevent the secretion of the cephalospinal fluid.

"The resorption of the cephalospinal fluid can be increased:

"a) The C.S. fluid leaks somewhere else — perforation of the dura.

"b) The blood osmotic pressure increases and allows an influx of the cerebro-spinal fluid into the circulation."

We would like to impress that the problem of intracranial hypotension is a particular case of water metabolism in the tissues, and bound to the problem of edema. This question is /473
the subject of many studies at this point, and we recommend the work of Mach on the states of dehydration which reviews the data, still incomplete, acquired to this day on the subject. This problem is complicated by a mechanical problem since the brain functions in the inextensible bony skull.

In our patients, where death seems imminent, the direct action on the troubles of blood circulation, even if they are primary (cardiovascular medication, operation on the sympathetic nervous system) is a mediocre help. On the other hand, the neurosurgical operations whose purpose is to regulate directly the aqueous balance in the brain and surrounding membranes are often efficient: these interventions which represent first an emergency neurosurgery have then a repercussion on the troubles of the blood circulation.

To end our exposé, we will insist on the fact that all the cerebromeningeal hydraulics are ruled by *the notion of equilibrium*, primordial phenomena already emphasized by Claude Bernard.

The tension of the cephalospinal fluid is a biological constant dependent upon many factors, vascular, mechanical, peripheral, and central nervous.

The normal functioning of the brain requires a certain equilibrium of the tension of the cephalosphinal fluid. If this balance is broken, the brain does not have too many modalities to express its suffering, either in the case of accident of hypertension (cerebral edema, serous meningitis, ventricular hydrocephaly), or of intracranial hypotension.

As we have seen, the analogy is great between those two states which can follow each other, or be associated to various degrees in the same patient.

Similar facts are also observed in general pathology, glycemia or calcemia, for instance, are also constants which can be submitted only to minor variation in the physiological state. An increase or a decrease in the blood sugar beyond certain limits brings about pathological disturbances.

Hyperglycemia or hypoglycemia can occur successively in the same patient, and the frequency is well known of hypoglycemia accidents in the diabetic when submitted to excessive doses of insulin.

As far as the notions of intracranial hypertension and hypotension are concerned, it is most important, for the therapy, when death becomes imminent, to recognize the dominant trouble in cerebromeningeal hydraulics in order to treat it correctly:

— drainage of the serous meningitis

— cerebromeningeal dehydration in cerebral edema

— ventricular equilibration in hydrocephaly

— rehydration and re-inflation of the ventricle in hypotension.

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